physician. Maldistribution is a complex issue which is better defined by each community's assessment of its own supply rather than relying on census figures or similar determinations.

As a participant in Ohio's Preferred Placement Program (PPP), I support the concept to finance medical education in return for future services in both rural and urban underserved areas. PPP has developed a financial instrument ($1.5 million) whereby local communities raise private sector monies to finance a student's medical education in return for practice obligations. The physician glut predicted by the Graduate Medical Education National Advisory Committee (GMENAC) encourages the dwindling of government subsidies for medical education. Whether this will filter down to a surplus of physician services for urban and rural underserved communities has yet to be determined. Organized medicine should support and lobby for programs that organize private sector monies for students both to insure equal socioeconomic access to medical education and to help curb maldistribution.

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Oral Contraceptives and Venous Thrombosis

TO THE EDITOR: In his article in the journal[1] on the systemic effects of oral contraceptives, Dr Thomas Kelly states, "In contrast to the controversial relationship between oral contraceptives and arterial occlusive disease, the data regarding the effect of their use on venous thrombosis are quite straightforward." Indeed, they are straightforward; but the scientific interpretation is quite different from that given by Dr Kelly. In a review published in 1973,[2] I pointed out that the association between ingestion of oral contraceptives and venous thrombosis found in retrospective studies could be accounted for by the wide publicity given previously in the medical and lay press of a possible risk of venous thrombosis in oral contraceptive users. As a result, any woman taking the pill and presenting with leg or chest pains would have been carefully examined for evidence of venous thrombosis or pulmonary embolism and many would have been admitted early to a hospital for further studies, such as venography. On the other hand, similar symptoms in a woman not taking the pill might have been dismissed by the patient or her physician as being merely due to leg cramps.

This hypothesis of a greater index of suspicion of venous thrombosis in women using the pill than in nonusers was subsequently supported by the studies of Barnes and coworkers in 1977.[3] They found that the incidence of venous thrombosis proved by Doppler examination in women taking oral contraceptives in whom venous disease was suspected was only 16.7% which was about half the incidence documented by Doppler in women not taking the pill. Furthermore, most prospective studies have not shown an increased incidence of venous thrombosis in women taking the pill. Indeed Dr Ramcharan, the principal investigator in the very large and well-controlled Walnut Creek study, concluded at the end of the study that oral contraceptives as a cause of thromboembolism had not been proved since the effect of diagnostic bias was a strong possibility. Moreover, a cause-and-effect relationship cannot be postulated merely on the basis of a statistical association.

That oral contraceptives induce an increase in certain clotting factors and a slight decrease of antithrombin III is not in dispute. However, no worker with any recent experience in the field of blood coagulation would any more deduce from these facts that these changes tip the hemostatic balance towards thrombosis than they would postulate that a lengthening of the clotting time necessarily indicates hypocoagulability. The facts are now very clear. But although there is no proof that contraceptives predispose towards venous thrombosis, it remains the general belief that a cause-and-effect relationship has been established and this is repeated from one textbook to another without any critical evaluation of the data on which the original claim was based.

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Dr Kelly Replies

TO THE EDITOR: Dr Hougie suggests that the preconceived impressions of epidemiologists who studied oral contraceptive effects caused them to intensively investigate symptomatic users of oral contraceptives and thus find a spurious association of those agents and venous thrombosis. The designers of early British studies of oral contraceptives were well aware that bias could influence retrospective, case-control studies, and they thoroughly discussed in their reports why bias was unlikely to explain the results they had obtained.[1] In particular, they found that diagnostic studies for venous thromboembolic disease were not employed more frequently in patients using oral contraceptives than in control subjects.[2]

To support his argument that physicians more readily study for thromboembolic disease women receiving oral contraceptives, Dr Hougie references a report in which women referred for Doppler evaluation of leg veins were only half as likely to have confirmation of thrombosis if they were taking oral contraceptives than if they were not. That result, however, is not a universal finding as others have reported observing no difference in the percent of venograms positive for deep venous thrombosis when young women with clinically suspected disease were divided according to oral contraceptive use. Tibbutt found that about twice as many oral contraceptive users were referred for study and at least twice as many had documented thromboses.[3]

Besides retrospective studies, recent prospective studies have also found an association of oral contraceptives and venous thrombosis.[4] Even the Walnut Creek study to which Dr Hougie refers found that oral contraceptive use was associated with an increased risk of idiopathic thromboembolism—that is, thrombosis without a recent surgical procedure, trauma, malignant condition or other known predisposing factor.[5] Some investigators feel that this evidence is weakened by the study's failure to find a statistically significant effect in patients predisposed to venous thromboembolism. They argue that an adverse effect of oral contraceptives should be most pronounced in patients at high risk for thrombosis. However, it can also be argued that other predisposing factors, when present, overwhelm the effect of oral contraceptives and prevent a small but real effect of those agents from being discernible.

In addition to the results of epidemiologic studies, much indirect evidence indicates an association of oral contraceptives and thromboembolic disease. High-dose, noncontraceptive estrogen therapy, such as for suppression of postpartum lactation, has been shown to increase the incidence of thromboembolism.[6] Furthermore, the decreased morbidity from venous thromboembolic disease in reproductive-age women that occurred coincidently with the introduction of contraceptive pills with reduced estrogen content[7] also implicates estrogen as a risk factor for deep venous thrombosis. Finally, the dose-related effect of oral contraceptives to create an apparent imbalance of the hemostatic mechanism towards hypercoagulability, while of uncertain physiologic significance, is consistent with the reported correlation between estrogen dose and the risk of venous thromboembolism.[8]

Thromboembolic events in all young women are infrequent, and so a conclusive answer regarding their incidence in oral contraceptive users as compared with nonusers is unlikely because of the enormous size required of a definitive study. While there is no undeniable proof for a causal relationship between oral contraceptives and venous thrombosis, there is a wealth of evidence for the association of the two which, after reviewing, I and many others have chosen to accept rather than disregard as the result of bias. Yet, I would emphasize that since the risks of oral contraceptive use are small and further reduced by minimizing the steroid content of the preparation used, oral agents are a wise choice for many women who want contraception.

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Relationship Between Costs and Quality of Medical Care

To the Editor: Your editorial in the August issue dealing with the measurement of quality and costs of medical care[1] reminded me of an interesting proposed relationship between the two. As a student several years ago in the Community Medicine program at the University of Utah School of Medicine I was exposed to the following equation:

$$\text{Quality of Medical Care} = \frac{\text{Outcome of Medical Care}}{\text{Cost of Medical Care}}.$$

Perhaps it was my undergraduate training in mathematics that predisposed me to "latch on" to this equation in such a complete way. Ever since, I have not been able to read an article or editorial dealing with costs and quality without thinking in terms of this relationship.

The beauty of such an expression is that it defines an admittedly "soft" or subjective variable (quality) in terms of two "hard" or more objective variables (outcome, cost). The outcome of a medical care event may be considered in terms of health or functional status, years of life saved or whatever. Costs may include direct costs only or might also include the indirect costs associated with the provision or consumption of that care. To the extent that one increases the value of the numerator while holding the denominator constant or, alternatively, decreases the denominator while holding the numerator constant, quality increases.

I must say, however, that I have always been troubled by my inability to "play" with this equation. For example, if one attempts to move cost from the denominator of the right-hand expression and place it in the lefthand expression what results is as follows: cost · quality = outcome. If one holds quality constant and increases cost, does outcome necessarily increase (improve)? Then if one wishes to move quality into the denominator of the right hand expression (by dividing each side by quality), what results is $\text{cost} = \frac{\text{outcome}}{\text{quality}}$. If one then holds outcome constant and increases quality does cost necessarily go down?

At any rate, given the importance of understanding the relationships between costs of medical care and quality of medical care, we might all benefit from adding this perspective to our conceptual armamentarium.

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Enterococcal Endocarditis After Sigmoidoscopy

To the Editor: The article by Rodriguez and Levine[1] in the June 1984 issue was timely, indeed. A patient of mine had just been admitted to Santa Monica Hospital with enterococcemia. She was a 67-year-old woman with a porcine mitral valve prosthesis for rheumatic heart disease, a pacemaker and multiple other medical problems.